revascularization (TLR), nonfatal myocardial infarction, and death at 6 years, after treatment with sirolimus-eluting stent (SES) of comparable unselected lesions.
Methods: A total of 1083 lesions in 994 patients undergoing SES implantation and subsequent coronary angiography 6 to 12 months after the index procedure were analyzed. SF was defined as a complete separation of stent segments or single or multiple stent strut fracture, as assessed by coronary angiography, plain fluoroscopy and/or intravascular ultrasound at follow up.
Results: At 6- to 12-month angiographic follow-up, SF was present in 90 of 1083 lesions (8.3%), and 89 of 994 patients (9.0%). In-stent late loss was significantly higher in SF lesions vs. non-SF lesions (0.48±0.62 mm vs. 0.13±0.50 mm, P<0.001), resulting in a significantly higher in-stent restenosis rate (20.0% vs. 4.4%, P<0.001). At 6 years, SF vs. non-SF patients had significantly higher rate of composite major adverse cardiac events (MACE) (27.0% vs. 17.7%, P=0.031), mainly driven by significantly higher TLR (23.6% vs. 13.6%, P=0.011) rate. Adverse effects of SF on clinical outcomes occurred mostly within the first year (16.9% vs. 7.3%, P=0.002), with similar MACE rate between 1 and 6 years (10.1% vs. 10.4%, P=0.935). No significant differences between SF vs. non-SF patients were observed in the cumulative frequency of early ST (0% vs. 0.6%, P=0.625), late ST (1.1% vs. 0.3%, P=0.313), very late ST (2.2% vs. 2.5%, P=0.281) or death/nonfatal myocardial infarction at 6 years (6.7% vs. 6.2%, P=0.837). In addition, there were no statistically differences in rates of early cessation of thienopyridine therapy within 6 months after the index procedure, at 19.8% in SF patients and 12.7% in non-SF patients (P=0.066).
Conclusions: The present study suggests that although SF patients have a higher MACE rate at 6 years compared with non-SF patients, that the increases in the rates of the events between years 1 and 6 are low and not significantly different between the 2 groups.

P5494 | BENCH
Would pfo closure prevent recurrent cryptogenic neurological events?

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Background: The prevalence of patent foramen ovale (PFO) among patients with cryptogenic stroke or transient ischemic attack (TIA) is higher than that in the general population. Closure is often recommended in such patients, but it is not known whether this intervention really reduces the risk of recurrent neurological events (RNE).
Objectives: The purpose of this study was to conduct a meta-analysis to evaluate the risk of RNE after PFO closure or medical therapy in patients with cryptogenic stroke or TIA due to presumed paradoxical thromboembolism.
Search methods: Two independent reviewers searched MEDLINE and extracted data from observational and randomized controlled trials comparing PFO closure and medical therapy to prevent RNE. Intention-to treat analysis was used to extract data.
Data collection and analysis: RevMan 5.2 (Nordic Cochrane Center, Copenhagen, Denmark) was used to pool the results of individual studies. The random effects model was applied. The effect size was presented using the risk ratio (RR). Statistical heterogeneity was evaluated using the I^2 statistic.
Results: Thirteen comparative observational and 3 randomized studies were identified in this analysis, including 2263 patients treated with PFO closure (94% using percutaneous device) and 2789 patients who received medical therapy. Overall, PFO closure reduced the number of patients with RNE by 50% (RR 0.50, 95% confidence interval [CI] 0.29–0.86, p = 0.01), with statistically significant heterogeneity among the studies (I^2 = 70%, p < 0.0001). In comparative observational studies, closure was superior to medical therapy (RR 0.40, 95% CI 0.20-0.81; p = 0.01). The incidence of events for the control arm of the randomized trials trial was lower than the summary estimate from observational studies; thus, there was no significant benefit of closure over medical treatment (RR 0.86, 95% CI 0.56-1.32; p = 0.49).
Conclusions: Although further randomized trial data are needed to precisely determine the effects of closure on stroke or TIA recurrence, the results of available randomized controlled trials challenge the credibility of a substantial body of observational evidence strongly favoring mechanical closure over medical therapy in secondary prevention of cryptogenic neurological events.

P5495 | BEDSIDE
The additional effect of Eicosapentanoic acid on coronary plaque stability in stable angina patients with statin use by Optical Coherence Tomography analysis

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Purpose: Statin treatment is demonstrated to reduce cardiovascular events. However, even intensive statin treatment remains residual coronary risk. Eicosapentanoic Acid (EPA)has promising benefits for second prevention. The aim of this study is to access the additional effect of EPA on coronary plaque stability in patients with statin use by using Optical Coherence Tomography (OCT).
Methods: Consecutive 14 patients who had received statin treatment before the past 6 month at baseline study with lipid-rich plaques on non-culprit lesion by OCT analysis were enrolled in this study. Of these,9 patients (10 lesions) received with EPA 1800mg/day after baseline study and 5 patients (5 lesions) with control group were assigned randomly. We measured thinnest Fibrous Cap Thickness (FCT) and the arc of lipid plaque at baseline and 9 month follow-up.
Results: The change of Serum Eicosapentanoic Acid/Arachidonic acid (EPA/AA) ratio were 0.31±1.08 at baseline, 1.08±0.38 at 9 month in EPA group (p=0.0001) and 0.24±0.13, 0.26±0.16 in control group (p=0.63) respectively. Patients with both of groups had significantly thick FCT (140±48.3μm, 246±80.4μm, p=0.0004 in EPA group and 152±26.8μm, 178±19.2μm, p= 0.04, in control group), however the difference in the change of FCT was highly significant in EPA group (106±19.2μm vs. 26±9.2μm, p=0.0003).The change of the arc of lipid plaque also reduced significantly in EPA group compared to control group (-40.0±11.5° vs. -4.0±5.48°, p=0.0074).

Table 1. Serum EPA/AA and OCT analysis

	EPA group (10 lesions, n=9)	Control group (5 lesions, n=5)	P value
EPA/AA	1.08±0.38	0.26±0.16	0.0009
Change of fibrous cap thickness (μm)	106±19.2	26±9.2	0.0003
Change of the arc of lipid plaque (degree)	–40.0±11.5	–4.0±5.48	0.0074

Conclusion: Additional EPA treatment was effective on coronary plaque stabilization with increasing fibrous cap thickness and decreasing the arc of lipid plaque.

INFLAMMATION AND MICROCIRCULATION

P5497 | BEDSIDE
Association of platelet reactivity and endothelial function and anti-inflammatory effects in patients with stable coronary artery disease

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Background: Platelets are actively involved in endothelial dysfunction and vascular atherosclerosis. We aimed to investigate the relationship between High On-treatment Platelet Reactivity (HOPR) and endothelial function of the peripheral arteries.
Methods: Ninety-one patients who planned to take follow-up angiography were consecutively enrolled. All patients were treated with 100mg aspirin and 75mg clopidogrel for at least 6 month due to prior coronary stent implantation. High-sensitivity C-reactive protein (hsCRP) was examined before coronary angiography. HOPR was defined as maximal platelet aggregation by 5 μmol/L of ADP > 50% by light transmittance aggregometry. Flow-Mediated Dilation (FMD) of the brachial artery was assessed using ultrasonography. Brachial-ankle pulse wave velocity (PWV) was measured using an automated device.
Results: There was no difference in baseline characteristics and previous coronary intervention data between ADP-induced platelet aggregation ≤ 50% (group 1, n=59) and HOPR (group 2, n=32). Group 2 showed significantly higher hsCRP levels than group 1. In group 2, significantly lower FMD (6.1±4.1 vs 12.9±6.2%) and higher PWV (1925.4±362.2 vs 1571.0±306.5 ms) values were noted compared with group 1 (Figure). Platelet reactivity showed negative correlation with FMD and positive correlation with PWV. In follow-up angiography, repeated coronary intervention was more performed in patients with HOPR.

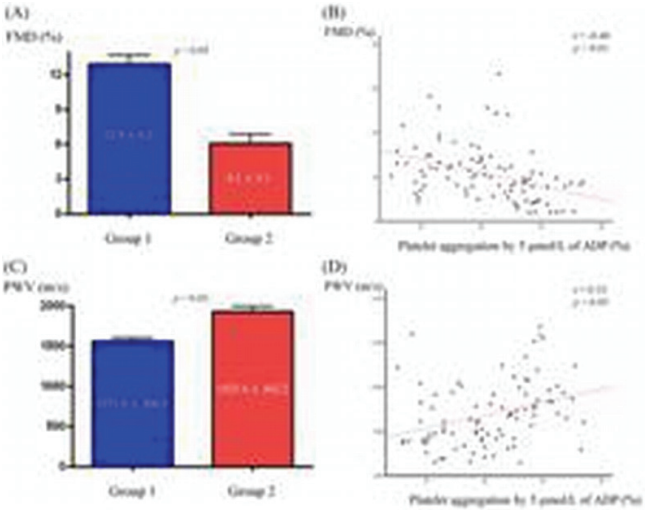

Figure 1

Conclusions: Inflammation, endothelial dysfunction and recurrent ischemic events were significantly associated with HOPR in patients under chronic clopidogrel treatment.